REMARKS/ARGUMENTS

By this amendment, claims 1-13 are amended. No new matter has been introduced. Claims 1-17, and 24-28 are pending. Reconsideration and allowance of all pending claims are respectfully requested.

Election/Restrictions

Non-elected claims 18-23 had been canceled in the communication filed April 3, 2003.

Priority

This application claims the priority of U.S. Provisional Patent Application No. 60/200,777, filed April 27, 2000. Applicants have amended the specification to include a reference to the prior application.

Drawings

Applicants have elected to use black and white drawings as acceptable drawings. A set of black and white drawings is attached.

Claim Objections

Claims 1-17 and 24-28 are objected to because of the informalities stated on pages 3 and 4 of the Office Action. Claims 1-17 have been amended to correct the informalities.

Rejections under 35 U.S.C. §102

Claims 1 and 2 stand rejected under 35 U.S.C. §102(b) as being anticipated by Koenig et al. Applicants respectfully traverse the rejection.

Koenig teaches the complete sequence of the human dystrophin cDNA. Claims 1 and 2, as amended, are directed to isolated nucleotide sequences comprising a dystrophin minigene encoding a protein consisting of a N-terminal domain; four to six rod repeats, an H1 domain of a dystrophin protein and an H4 domain of the dystrophin protein, a cysteine-rich domain, and, in the case of claim 2, the last three amino acids of a C-terminal domain of the dystrophin protein. The claimed dystrophin minigenes are substantially smaller than the full-length dystrophin gene but are capable of ameliorating dystrophic pathology when expressed in a dystrophic muscle. Accordingly, Applicants respectfully submit that claims 1 and 2 are not anticipated by Koenig. Withdrawal of the 35 U.S.C. §102(b) rejection is respectfully requested.

Claims 8-13 and 24–28 stand rejected under 35 USC 102(a) as being anticipated by Takeda. Takeda generally describes several mini-dystrophin genes having 4.5 kb or less but does not show any valid evidence that the minigenes are functional in vivo. The Examiner cited the article by Wang which alleges that "the mini-dystrophin genes tested by Takeda were functionally similar to CT dystrophin construct, thus sufficient to restore DAP complexes." Applicants respectfully traverse the rejection.

Applicants respectfully submit that restoration of DAP complexes does not necessarily result in the ameliorating dystrophic pathology, as claimed by the present invention. It has been demonstrated that restoration of DAP complexes in mdx mice

using non-muscle dystrophin gene restore the DAP complex but fails to prevent dystrophic symptoms (See Cox et al., Nature Gent. 8:333-339, 1994; Greenberg et al., Nature Gent. 8:340-344, 1994; and the review article by Eric Hoffman, Nature Gent. 8:311-312, 1994). In fact, the sentence cited by the Examiner from Wang's article specifically addresses this issue. The full sentence reads "the mini-dystrophin genes tested by Yuasa and colleagues (i.e. Takeda's group), although containing both intact N- and C-terminal domains and 1-3 central rod repeats, were functionally similar to CT dystrophin construct (Dp71), thus sufficient to restore DAP complexes **but insufficient to restore myofiber morphology and prevent dystrophic pathology."**

In addition, Applicants have amended claims 8-13 as suggested by the Examiner. Accordingly, Applicants respectfully submit that Takeda does not anticipate the amended claims 8-13, as well as claims 24-28, which depend from claims 9-13, respectively. Withdrawal of the 35 U.S.C. 102(a) rejection is respectfully requested.